Health Consultation

Evaluation of Contaminants: Residential Domestic Well near the Hamilton/Labree Road PCE Site
Hamilton Road PCE
Chehalis, Lewis County, Washington

August 16, 1999
Contact information revised February 26, 2004

Prepared by
Washington State Department of Health
under cooperative agreement with the
Agency for Toxic Substances and Disease Registry

Washington State Department of Health

FOREWORD

The Washington State Department of Health (DOH) has prepared this health consultation under cooperative agreement with the Agency for Toxic Substances Disease Registry (ATSDR), an agency of the U.S. Department of Health and Human Services. The goal of the DOH and ATSDR is to identify and mitigate adverse human health effects resulting from exposure to hazardous substances in the environment. This report was prepared in accordance with methodologies and guidelines developed by ATSDR.

A health consultation provides advice on specific public health issues which may arise as a result of an actual or potential human exposure to a hazardous substance. Health consultations provide a means for DOH to respond quickly to a request for health information on hazardous substances and to make recommendations for actions to protect public health. DOH evaluates available information about hazardous substances at a site, determines whether exposures have occurred or could occur, and reports the potential harmful effects from exposure.

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BACKGROUND AND STATEMENT OF ISSUES

The Lewis County Department of Public Health (LCDPH), through a request from a resident (petitioner), asked the Washington State Department of Health (DOH) to evaluate potential health risks from exposure to tetrachloroethylene (PCE) detected in the petitioner's domestic well. The well is in the vicinity of the Hamilton/Labree Roads PCE site, approximately three miles south of Chehalis, in Lewis County, Washington. This health consultation summarizes DOH's evaluation of the public health implications from exposure to contaminants in this well.

In late 1993 and early 1994, DOH testing of local drinking water wells revealed that some shallow wells were contaminated with PCE at concentration ranging from 3 µg/l to 2,165 µg/l. LCDPH and the Washington State Department of Ecology (Ecology) informed affected well owners of the test results and advised them to obtain alternate sources of drinking water. In December 1996, DOH prepared a health consultation which evaluated PCE-contaminated water supplies for 2 businesses and 3 residences near Chehalis [3]. Ecology is currently supplying bottled drinking water for some of the families and businesses in the affected area, including the petitioner's. Under the authority of the Washington State Model Toxics Control Act, Ecology has identified two probable PCE source areas in the Hamilton/Labree Road area. The first source is believed to be in the area northeast of the Hamilton/Labree Roads intersection, and the second source is believed to be in the vicinity of a painting facility located between Hamilton Road and the Berwick Creek, southeast of the first source (Figure 1). Volatile organic compounds (VOCs) have been detected in the shallow aquifer in the vicinity of the source areas, and include tetrachloroethylene (PCE), trichloroethylene (TCE), cis and trans-1,2-dichloroethene, and vinyl chloride [1]. Ecology recently installed seven new monitoring wells near the second source area to gather additional groundwater information intended to aid in selecting appropriate source control measures. In July 1999, Ecology plans to collect additional water samples from the petitioner's kitchen sink tap and from six previously sampled domestic wells, as well as from the seven new monitoring wells and eight existing monitoring wells.

The petitioner moved to her current residence, a mobile home, in September 1996, and has lived there ever since. She became pregnant in November 1996, and delivered in August 1997. The petitioner was pregnant during the time she was exposed to PCE. The petitioner was exposed to PCE dermally, and through ingestion and inhalation routes from September 1996 until October 1997 (13 months). From October 1997 to March 1999 (17 months), the petitioner used bottled water supplied by Ecology for drinking and cooking purposes, but was still exposed to PCE dermally and through inhalation of PCE vapors from the shallow well from showering and other hot water uses. Exposure to PCE was eliminated when the petitioner began receiving water from the deep aquifer well in March 1999[1]. The petitioner continues to use bottled water for drinking and cooking purposes, and uses water from the deep aquifer well for non-consumptive purposes,

[1]The low detection of PCE from the petitioner's kitchen sink tap in February 1999 does not appear to be a product of the deep aquifer since no PCE was detected in the associated deep wellhead sample. The source of this detection is believed to be located between the wellhead and the petitioner's kitchen sink tap, possibly due to residual contamination.

such as dishwashing and showering.

Since approximately February 1998, the petitioner indicated she and/or members of her family have suffered from gastrointestinal (diarrheal episodes) and respiratory problems, as well as short-term skin problems (itching, redness, dryness, etc.) [5].

Between the Fall of 1993 and February 1999, a total of nine samples were collected from well 5, three by the DOH Drinking Water Division, one by a Department of Ecology consultant, and six by the Department of Ecology (Table 1). The initial eight samples were tested only for volatile organic compounds (VOCs). The February 1999 sample was tested for VOCs, semi-volatile organic compounds (SVOCs), pesticides, herbicides, metals, and conventional parameters (total organic carbon, temperature, nitrate, pH, alkalinity, chloride, total dissolved solids, dissolved oxygen, sulfate, etc.). ***PCE was detected in all nine samples, and has been the only contaminant detected above a health-based comparison value (Table 1)***.

Detections of PCE at the well 5 wellhead have ranged from 3.3 µg/l to 11 µg/l. The most recent (February 1999) sample collected at the petitioner's kitchen sink tap showed a PCE concentration of 0.24 µg/l. The source of this detection is suspected to be from between the wellhead and the petitioner's kitchen sink tap as no PCE was detected at the wellhead during the same sampling event. Prior sampling of the petitioner's current water source (at the deep well wellhead) has not shown any contamination. The most recent analysis of water from the shallow well 5 wellhead showed a PCE concentration of 7.1 µg/l. A chronology of all detected contaminants, corresponding concentrations, and health-based comparison values are listed in Table 1.

TABLE 1
DOMESTIC WELL CONTAMINANT CONCENTRATIONS
WELL 5
(October 1993 - February 1999)

Sample Collection Date	Owner/Depth	Sample Location	Analyte	Concentration (µg/l)	❊Carcinogenic Comparison Value (µg/l)	★Non-carcinogenic Comparison Value (µg/l)	MCL (µg/l)
October 1993[1]	Veenhouwer/Shallow		PCE	*3.3*	0.7	100	5
June 1996[1]	Veenhouwer/Shallow		PCE	*5.8*	0.7	100	5
March/May '97[2]	Veenhouwer/Shallow		PCE	7	0.7	100	5
October 1997[3]	Veenhouwer/Shallow		PCE	*7.7*	0.7	100	5
January 1998[3]	Veenhouwer/Shallow		PCE	*7.6*	0.7	100	5
April 1998[3]	Veenhouwer/Shallow		PCE	*11*	0.7	100	5
July 1998[3]	Veenhouwer/Shallow		PCE	*7.9*	0.7	100	5
December 1998[3]	Veenhouwer/Shallow	A.N. Kitchen Sink Tap	PCE	*5.3*	0.7	100	5
December 1998[3]	Veenhouwer/Shallow	Wellhead	PCE	*4.7*	0.7	100	5
December 1998[3]	Veenhouwer/Shallow	O.N. / J.L. Kitchen Sink Tap	PCE	*5*	0.7	100	5
February 1999[3]	Veenhouwer/Deep	O.N. / J. L. Kitchen Sink Tap	PCE	0.24 (J)	0.7	100	5

Sample Collection Date	Owner/Depth	Sample Location	Analyte	Concentration (µg/l)	❊Carcinogenic Comparison Value (µg/l)	★Non-carcinogenic Comparison Value (µg/l)	MCL (µg/l)
February 1999[3]	Veenhouwer/Deep	Wellhead	PCE	ND	0.7	100	5
February 1999[3]	Veenhouwer/Shallow	A.N. Kitchen Sink Tap	PCE	*6.3*	0.7	100	5
February 1999[3]	Veenhouwer/Shallow		Copper	7.3	NA	NA	1,300
February 1999[3]	Veenhouwer/Shallow		Zinc	5.7	NA	3,000	NA
February 1999[3]	Veenhouwer/Shallow		Nitrate	2,500	NA	16,000	10,000
February 1999[3]	Veenhouwer/Shallow		PCE	*7.1*	0.7	100	5

µg/l = micrograms of chemical per liter of water (equals one part per billion)
★Non-carcinogenic Comparison Value = ATSDR child RMEG (Reference Dose Media Evaluation Guide)
❊Carcinogenic Comparison Value = ATSDR CREG (Cancer Risk Evaluation Guide)
MCL = Federal Safe Drinking Water Act Maximum Contaminant Level
NA = Not available
ND = Not detected
shaded/*italicized* value = concentration which exceeded an ATSDR health-based comparison value that required further evaluation
[1] = Sampled by DOH
[2] = Sampled by Ecology's contractor, SAIC
[3] = Sampled by Ecology

DISCUSSION

ATSDR has developed health-based comparison values for chemicals in various media, including water, which define a concentration at or below which carcinogenic and/or noncarcinogenic health effects are unlikely to result from exposure. Contaminant concentrations exceeding these values do not necessarily pose a health threat, but are further evaluated to determine potential health effects. As PCE in the petitioner's well exceeded an ATSDR comparison value, it was further evaluated to determine whether health effects are likely. Since exposures (through either ingestion and/or inhalation and dermal routes) occurred between September 1996 and February 1999, estimated doses used to evaluate health risks assumed a 30 month exposure duration (13 months through ingestion, inhalation, and dermal routes, and 17 months through only inhalation and dermal routes). During this time period, PCE concentrations ranged from 4.7 µg/l to 11 µg/l, with a mean concentration of 7 µg/l (Table 1).

Evaluating non-cancer risk

Although concentrations of PCE were well below the ATSDR non-cancer comparison value, because of the petitioner's specific health concerns, DOH evaluated available literature to assess the potential for health effects. In estimating exposure doses, it was assumed that the petitioner and her family were exposed to the mean (7 µg/l) detected PCE concentrations in their drinking water well during the exposure period.[2] Estimated doses were then compared to the EPA oral reference dose (RfD). RfDs are estimates of daily exposure of a human to a chemical that is likely to be without an appreciable non-cancer risk over a chronic (long-term) exposure duration. They are derived from toxic effect levels obtained from human and laboratory animal studies. The toxic effect levels are expressed as either the lowest observed adverse effect level (LOAEL) or the no-observed adverse effect level (NOAEL). In human or animal studies, the LOAEL is the lowest dose at which an adverse effect is seen, while the NOAEL is the highest dose that did not result in any adverse health effects.

To account for uncertainty (i.e.-intraspecies variability, interspecies variability and extrapolation of a subchronic effect level to its chronic equivalent)., the toxic effect levels are divided by safety factors (usually 100 or 1,000) to provide the more protective RfD. If a dose exceeds the RfD, the *potential* exists for adverse health effects. Thus, a dose only slightly exceeding the RfD is usually still well below the toxic effect level. The higher the estimated dose is above the RfD, the closer it will be to the toxic effect level.

Evaluating cancer risk

For screening of chemicals which are known or expected to cause cancer, it is assumed that no "safe" level exists, and EPA cancer slope factors are used to calculate an "estimated" increased

[2] DOH conservatively assumed exposure from all 3 routes (ingestion, inhalation, and dermal), although over half of the 30 month exposure duration was from only inhalation and dermal routes. As a result, estimated risk was overestimated.

cancer risk. An exposure which results in an estimated increased cancer risk of one additional cancer in a population of one million people exposed for thirty years, averaged over a 70 year lifetime, is considered an acceptable risk, and is used as the comparison value. In a population of one million men in the U.S., 333,000 (one in three) are expected to develop cancer from all causes in their lifetime (through 79 years of age). For U.S. woman, the figure is 200,000 [12]. The additional estimated cancer risk means that if those one million men are exposed for 30 years to this level of the chemical, 333,001 would be expected to develop cancer. For those one million woman exposed, 200,001 would be expected to develop cancer.

Contaminants exceeding a comparison value which were further evaluated

The following contaminant exceeded an ATSDR health-based comparison value and was further evaluated in the health consultation:

Tetrachloroethylene (PCE)

PCE is a manufactured compound widely used for dry cleaning fabrics and as a metal degreaser. It is also used as an intermediate in the manufacturing of other products [2]. Cancer and noncancer toxicity is discussed below.

Non-cancer Effects

Liver and kidney damage have been observed in laboratory animal studies after exposure to high doses of PCE. Liver weight/body weight ratios were significantly higher than controls for animals treated with 100 mg/kg/day of PCE. At higher doses, hepatotoxic effects included decreased DNA content, increased SGPT, decreased levels of G6P and hepatocellular necrosis, degeneration and polyploidy [2,6].

Groups of 20 Sprague-Dawley rats of both sexes were administered doses from 20,000-2,000,000 times greater than those estimated for the petitioner. Males in the high-dose group and females in the two highest groups exhibited depressed body weights. Equivocal evidence of hepatotoxicity (increased liver and kidney weight/body weight ratios) were also observed at the higher doses [2,6].

Relative sensitivity to man cannot be readily established, but the RfD of 0.01 mg/kg/day is protective of the most mild effects observed in humans [diminished odor perception/modified Romberg test scores in volunteers exposed to 100 ppm for 7 hours; roughly equivalent to 20 mg/kg/day].

As the petitioner's estimated exposure dose was 25 times lower than the oral RfD, ***noncancer health effects are unlikely to result from exposure***. The RfD was derived from a NOAEL of 14 mg/kg/day, and a LOAEL of 71 mg/kg/day, based on hepatotoxicity in mice and weight gain in rats.

Cancer Effects

Various case-control studies were evaluated for possible associations between exposure to PCE and cancer effects in human populations. Although some of these studies indicate a possible association between exposure to PCE and various cancers, including bladder cancer, kidney cancer, and leukemia, the studies had limitations which precluded definitive conclusions.

Cancer has been reported in experimental animals after oral exposure to PCE. Statistically significant increases in hepatocellular carcinomas occurred in the treated mice of both sexes. A cancer effect level (CEL) of 386 mg/kg/day was derived from a chronic mouse study [2]. The cancer effects in this study were hepatocellular carcinomas. ***However, the petitioner's estimated exposure dose was 965,000 times lower than this CEL.***

An EPA workgroup is currently reassessing PCE carcinogenicity, so has removed the oral slope factor. Completion of the reassessment, which was announced in the January 2, 1998 Federal Register notice, is planned for FY 1999 or FY 2000 [6]. In 1987, an EPA carcinogen assessment proposed PCE as a probable human carcinogen. In light of new data, EPA reviewed findings that suggest the weight-of-evidence for PCE as a possible human carcinogen - probable human carcinogen continuum. Presently, the agency has not adopted a final position on the classification of human carcinogenicity for this chemical [3].

In order to estimate the cancer risk from exposure to PCE, the former oral slope factor was used. ***The estimated increased cancer risk, assuming a continuous 30 month (2.5 year) exposure to the mean concentration (7 μg/l) of PCE in drinking water from well 5, is insignificant; approximately 7 additional cancers in a population of 10,000,000 persons exposed, averaged over a 70 year lifetime.*** The actual risk is likely even less than this since this estimated exposure dose assumed inhalation, dermal, *and ingestion* routes of exposure for the entire 30 month time period. For part of this time, however, exposure occurred only through inhalation and dermal routes, not from ingestion.

SPECIFIC HEALTH CONCERNS

As previously noted, the petitioner's health concerns relate to the gastrointestinal and respiratory tract, as well as the skin [5,10]. The literature was reviewed to assess whether exposure to PCE at the levels detected could result in the effects noted above.

GASTROINTESTINAL EFFECTS

A 1937 study showed that boys exposed to unspecified oral doses of PCE to remove intestinal worms resulted in vomiting. In another study, histological changes in the gastrointestinal tract were not observed in rats or mice treated by gavage with PCE for 78 weeks at doses that increased mortality [2]. The studies appear to indicate that gastrointestinal health effects could result from exposure to PCE, but only at very high doses. Gastrointestinal effects would not be expected to result from exposure to the low PCE levels detected in the petitioner's well.

RESPIRATORY EFFECTS

Various inhalation studies have observed respiratory irritation from exposure to PCE, but at levels many thousands of times higher than would result from volatilization of PCE from the petitioner's well water. Exposure of mice to very high levels of PCE in the air resulted in an increased susceptibility to infection from two strains of bacteria [2]. Similarly, congestion of the lungs was reported in rats exposed to PCE at levels many thousands of times higher than levels expected to result from volatilization of PCE from the petitioner's well water. Respiratory effects would not be expected to result from inhalation of PCE vapors from the petitioner's well water.

DERMAL (SKIN) EFFECTS

Studies have shown dermal effects from exposure to PCE, but only when exposed to pure solution (i.e.-100% PCE). In a 1964 study, volunteers who had placed their thumbs in beakers of PCE all experienced a burning sensation within 10 minutes. Chemical burns characterized by severe cutaneous erythema, blistering, and sloughing have resulted from prolonged accidental contact exposure to PCE used in dry-cleaning operations. Rabbits did not develop skin problems after being exposed dermally to pure PCE [2]. The studies indicate that although dermal effects are possible from PCE exposure, the concentration necessary to produce these effects are much higher than PCE levels measured in the petitioner's well water.

CHILD HEALTH INITIATIVE: REPRODUCTIVE/DEVELOPMENTAL EFFECTS

ATSDR's Child Health Initiative recognizes that the unique vulnerabilities of infants and children deserve special emphasis with regard to exposures to environmental contaminants. Infants, young children, and the unborn may be at greater risk than adults from exposure to particular contaminants. Exposure during key periods of growth and development may lead to malformation of organs (teratogenesis), disruption of function, and even premature death. In certain instances, maternal exposure, via the placenta, could adversely effect the fetus. After birth, children may receive greater exposures to environmental contaminants than adults. Children are often more likely to be exposed to contaminants from playing outdoors, ingesting food that has come into contact with hazardous substances, or breathing soil and dust. Pound for pound body weight, children drink more water, eat more food, and breathe more air than adults. For example, in the United States, children in the first 6 months of life drink 7 times as much water per pound as the average adult. The implication for environmental health is that, by virtue of children's lower body weight, given the same exposures, they often receive significantly higher relative contaminant doses than adults.

DOH evaluated the likelihood that infants and children living in the residences tested, are being exposed to PCE in drinking water at levels of health concern. The literature was reviewed on studies evaluating reproductive and developmental effects. ***Mean concentrations of PCE from well 5 samples were well below concentrations observed to result in adverse reproductive or developmental health effects***.

Reproductive effects in humans after oral exposure to tetrachloroethylene remains uncertain.

Lowest observed adverse effect levels (LOAELs) for reproductive effects were observed after acute oral exposures, and included significant increases in resorptions [2]. However, the levels at which these effects were observed were over 2 million times the petitioner's estimated exposure dose.

In an acute oral mouse study, developmental effects included increased activity [2]. However, the Minimal Risk Level (MRL) derived from this study was 125 times higher than the petitioner's estimated exposure. As a result, ***no reproductive or developmental effects are expected to result from exposure to tetrachloroethylene at the levels measured in the petitioner's well***.

RESULTS OF MEDICAL EVALUATION

Because of health concerns, Ecology and DOH assisted in scheduling a medical evaluation for the petitioner and her 2 children with Environmental and Occupational Health physicians at the University of Washington's Pediatric Environmental Health Specialty Unit. Ecology provided the physicians with all available exposure histories for the family prior to the medical evaluations. The evaluation occurred at Children's Hospital in Seattle on February 12, 1999. The physicians only evaluated the petitioner's children. The examinations did not include the collection of biological samples.

After the evaluation, the physicians concluded that, with regard to the skin, "physical examination of the sites excluded any serious condition." The physicians also believed the skin problems were unlikely a result of PCE exposure. Similarly, they concluded that it is extremely unlikely that the respiratory and diarrhea symptoms are related to exposure to PCE [5]. Since the medical evaluation, the petitioner has met with a pediatric gastroenterologist in Tacoma. The results of that visit have not been provided to DOH. DOH provided a copy of the medical evaluation summary letter to the petitioner.

CONCLUSIONS

After evaluation of the sampling data, *DOH concludes that there is no health threat to children or adult residents from past exposure to PCE in well 5 during the exposure period. As a result, there is no apparent public health hazard.* ATSDR uses the "*no apparent public health hazard*" category for sites where human exposure to contaminated media is occurring or has occurred in the past, but the exposure is below a level of health hazard.

Recommendations

1. The petitioner and her family should continue not to use shallow well 5 for domestic uses.

2. The petitioner's kitchen sink tap should be re-sampled to confirm the low PCE detection of February 1999.

3. Ecology, LCDPH, and the DOH Division of Drinking Water should continue to provide the DOH ATSDR Program the results of future domestic well samples collected from well 5, or any other domestic well in the vicinity of the Hamilton Road source areas for evaluation.

4. Upon request, DOH is available to provide further technical assistance to Ecology, the LCDPH, or the community, such as the evaluation of other affected water supplies, or to provide resources such as chemical-specific fact sheets or other related health effects information.

Appendix A - Exposure assumptions:

For this health consultation, it was assumed that residents were exposed 365 days per year, for the entire exposure period (2.5 years) to the mean PCE concentration (7 µg/l) in well 5. The petitioner was assumed to ingest 2 liters of water per day. Exposure was assumed to be through ingestion (drinking) and non-ingestion (inhalation and dermal) routes. Non-ingestion exposures were assumed to occur during household activities such as cooking, bathing, and dishwashing, and were assumed to be equal to exposures through ingestion.

Appendix B-Exposure formulas:

Exposure dose = [(C x IR x EF x ED)/BW x AT)] x 2

Estimated additional lifetime cancer risk = Estimated exposure dose x CSF

where:
C = concentration of contaminant (µg/l)
IR = ingestion rate (liters of water/day)
EF = exposure frequency (days/year)
ED = exposure duration (total # of years in exposure period)
BW = body weight
AT = averaging time (70 years x 365 days/year)
CSF = Cancer slope factor (estimates the excess upperbound lifetime probability of an individual developing cancer from an exposure to a carcinogen)

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GLOSSARY

MRL: ATSDR's Minimal Risk Level. An estimate of daily human exposure to a dose of chemical that is likely to be without an appreciable risk of adverse noncancerous health effects over a specified duration of exposure. MRLs are derived when reliable and sufficient data exist to identify the target organ(s) of effect or the most sensitive health effect(s) for a specific duration via a given route of exposure. MRLs can be derived for acute, intermediate, and chronic duration exposures by the inhalation and oral routes.

CREG: ATSDR's Cancer Risk Evaluation Guide. A concentration in air, water, or soil (or other environmental media), which is derived from EPA's cancer slope factor and carcinogenic risk of $1x10^{-6}$ for oral exposure. It is the concentration that would be expected to cause no more than one excess cancer in a million persons exposed over a lifetime.

RfD: An estimate (with uncertainty spanning perhaps an order of magnitude) of the daily exposure level of the human population, including sensitive subpopulations, to a potential hazard that is likely to be without an appreciable risk of deleterious effects (non-cancer) during a lifetime. It was developed to be protective for long-term exposure to a compound (7 years or longer).

CANCER SLOPE FACTOR: A plausible upperbound estimate of the probability of a response per unit intake of a chemical over a lifetime. The slope factor is used to estimate an upperbound probability of an individual developing cancer as a result of a lifetime of exposure to a particular level of a potential carcinogen.

LOAEL: Lowest Observed Adverse Effect Level. LOAEL's have been classified into "less serious" or "serious" effects. In dose-response experiments, the lowest exposure level at which there are statistically or biologically significant increases in the frequency or severity of adverse effects between the exposed population and its appropriate control.

NOAEL: No Observed Adverse Effect Level. The dose of a chemical at which there were no statistically or biologically significant increases in frequency or severity of adverse effects seen between the exposed population and its appropriate control. Effects may be observed at this dose, but were judged not to be "adverse".

MCL: Federal Maximum Contaminant Level. A drinking water regulation established by the Safe Drinking Water Act. It is the maximum permissible concentration of a contaminant in water that is delivered to the free-flowing outlet of the ultimate user of a public water system. MCLs are enforceable standards.

CARCINOGEN: Any substance that can cause or contribute to the production of cancer.

CONTAMINANT: Any substance or material that enters a system (the environment, human body, food, etc.) where it is not normally found.

MONITORING WELLS: Wells developed to collect groundwater samples for the purpose of

physical, chemical, or biological analysis to determine the amounts, types, and distribution of contaminants.

COMPARISON VALUE: A concentration used to select contaminants of concern at hazardous waste sites that are further evaluated in the health assessment process. The terms comparison value and screening level are often used synonymously.

MTCA: Model Toxics Control Act. Washington States hazardous waste cleanup law.

FIGURES

Figure 1: General Map of Chehalis, WA. And vicinity

Figure 2: Hamilton/Labree Roads Chlorinated Solvent Site Well Locations

Figure 3: Demographic site map, Hamilton Road PCE site, Chehalis, WA.